



ACC.15

TCT@ACC-12 | innovation in intervention

A1629
JACC March 17, 2015
Volume 65, Issue 10S

Stable Ischemic Heart Disease

IDENTIFICATION OF ASPIRIN RESPONSE RELATED GENE PROFILES IN THE ELDERLY POPULATION WITH CORONARY ARTERY DISEASE

Poster Contributions

Poster Hall B1

Saturday, March 14, 2015, 3:45 p.m.-4:30 p.m.

Session Title: Molecular Biology Pathways of Cardiovascular Disease

Abstract Category: 25. Stable Ischemic Heart Disease: Basic

Presentation Number: 1161-378

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Background: Aspirin is widely used in the primary and secondary prevention of cardiovascular diseases. Nevertheless, responses to aspirin vary from one patient to another. Recent studies showed aspirin exposure have influence on the expression of various genes, thus responsible for the variability in aspirin responses. The aim of our study was to identify aspirin response related gene profiles, and investigate the correlation between target genes expression and clinical outcomes in the elderly population with coronary artery disease (CAD).

Methods: A total of 160 aged patients with CAD treated with low-dose aspirin (100mg/d) were enrolled in this study. All enrolled patients were distributed according to quartile of 0.5mmol/l AA-induced platelet aggregation, and Aspirin Resistance (AR) was defined as the upper quartile of AA-induced platelet aggregation. Expression of fourteen genes (CLU CMT5 CTTN MPL TMEM64 SELP HLA-DQA1 HLA-DRB4 ITGA2B ITGB3 THBS1 CXCL5 PPBP SPARC) were measured using real-time quantitative PCR method.

Results: The quartile cut points of AA-induced platelet aggregation for the 25th, 50th, and 75th percentiles of the enrolled population were 9.48%, 12.16%, and 15.17%, respectively. AR was defined as AA-induced platelet aggregation $\geq 15.17\%$. Expressions of 5 genes (CTTN HLA-DQA1 HLA-DRB4 THBS1 ITGB3) were significantly higher in AR group than no-AR group ($P < 0.01$). Besides, with a mean follow-up of 6 months, clinical outcomes occurred more frequently in AR group as compared to no-AR group ($P < 0.05$). What's more, ten genes were differentially expressed in patients who experienced cardiovascular events during aspirin therapy, 3 up-regulated (CLU CMT5 CXCL5, $P < 0.05$) and 7 down-regulated (CTTN HLA-DQA1 ITGA2B MPL SELP SPARC TMEM64, $P < 0.05$) significantly.

Conclusion: Five genes were up-regulated in AR group when compared with no-AR group. Furthermore, higher risks of cardiovascular events and death were observed in AR group. In addition, expression of 7 genes were correlated with cardiovascular events during aspirin therapy. As a result, identification of these gene profiles might help distinguish patients with poor responses and susceptible to cardiovascular events.